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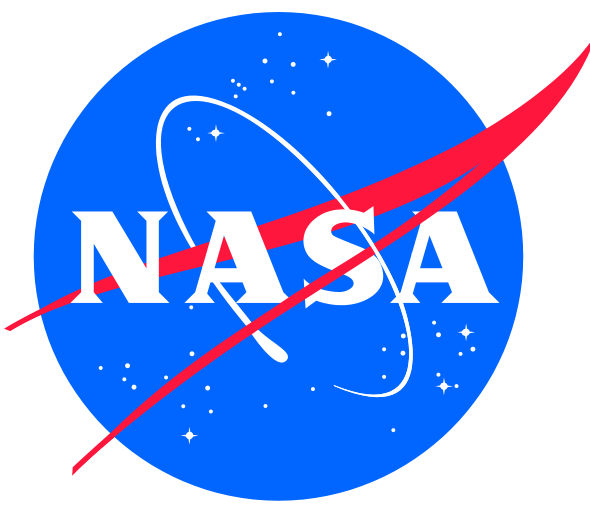
DECOMPRESSION SICKNESS AFTER AIR BREAK IN PREBREATHE DESCRIBED WITH A SURVIVAL MODEL

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BACKGROUND: Data from Brooks City-Base show the decompression sickness (DCS) and venous gas emboli (VGE) consequences of air breaks in a resting 100% O₂ prebreathe (PB) prior to a hypobaric exposure. **METHODS:** DCS and VGE survival times from 95 controls for a 60 min PB prior to 2-hr or 4-hr exposures to 4.37 psia are statistically compared to 3 break in PB conditions: a 10 min (n=40), 20 min (n=40), or 60 min break (n=32) 30 min into the PB followed by 30 min of PB. Ascent rate was 1,524 meters / min and all exposures included light exercise and 4 min of VGE monitoring of heart chambers at 16 min intervals. DCS survival time for combined control and air breaks were described with an accelerated log logistic model where exponential N₂ washin during air break was described with a 10 min half-time and washout during PB with a 60 min half-time. **RESULTS:** There was no difference in VGE or DCS survival times among 3 different air breaks, or when air breaks were compared to control VGE times. However, 10, 20, and 60 min air breaks had significantly earlier survival times compared to control DCS times, certainly early in the exposures. **CONCLUSION:** Air breaks of 10, 20, and 60 min after 30 min of a 60 min PB reduced DCS survival time. The survival model combined discrete comparisons into a global description mechanistically linked to asymmetrical N₂ washin and washout kinetics based on inspired pN₂. Our unvalidated regression is used to compute additional PB time needed to compensate for an air break in PB within the range of tested conditions.



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ABSTRACT

BACKGROUND: Data from Brooks City-Base show the decompression sickness (DCS) and venous gas emboli (VGE) consequences of air breaks in a resting 100% O₂ prebreathe (PB) prior to a hypobaric exposure. METHODS: DCS and VGE survival times from 95 controls for a 60 min PB prior to 2-hr or 4-hr exposures to 4.37 psia are statistically compared to 3 break in PB conditions: a 10 min (n=40), 20 min (n=40), or 60 min break (n=32) 30 min into the PB followed by 30 min of PB. Ascent rate was 1,524 meters / min and all exposures included light exercise and 4 min of VGE monitoring of heart chambers at 16 min intervals. DCS survival time for combined control and air breaks were described with an accelerated log logistic model where exponential N₂ washin during air break was described with a 10 min half-time and washout during PB with a 60 min half-time. RESULTS: There was no difference in VGE or DCS survival times among 3 different air breaks, or when air breaks were compared to control VGE times. However, 10, 20, and 60 min air breaks had significantly earlier survival times compared to control DCS times, certainly early in the exposures. CONCLUSION: Air breaks of 10, 20, or 60 min after 30 min of a 60 min PB reduced DCS survival time. The survival model combined discrete comparisons into a global description mechanistically linked to asymmetrical N₂ washin and washout kinetics based on inspired pN₂. Our unvalidated regression is used to compute additional PB time needed to compensate for an air break in PB within the range of tested conditions.

INTRODUCTION

Few data are available to understand the DCS and VGE consequences of an air break in an otherwise normal resting 100% O₂ PB, and none are available after PB that includes exercise.

DeHart (1) states, "Air-breathing interruptions of only a few min greatly decrease the efficacy of denitrogenation in the prevention of decompression sickness", but provides no references.

Estimates for O₂ PB payback time have ranged from one (2) to 35 times (3) the length of the break in PB. Payback time is the numbers of min of additional PB time needed to compensate for an interruption in the original PB time.

METHODS

We used an accelerated log logistic survival model accounting for asymmetrical N₂ washout and washin to describe DCS survival times in data where 0-min, 10-min, 20-min, or 60-min air breaks occurred at 30 min into a 60 min resting PB, including a six min ascent on 100% O₂.

Details about survival models and maximum likelihood optimization are available elsewhere (4-7).

Subjects ascended to 4.37 psia at 1,524 MPM to perform repetitive light exercise plus ambulation for 2 hrs (n = 95) or 4 hrs (n = 28) in the controls, and for 4 hrs in the three experimental conditions (n = 112).

VGE monitoring was every 16 min, for 4 min, using Hewlett-Packard SONOS 1000 Echo Imaging System with parasternal, short-axis view of the heart.

Our hypothesis is that N₂ washin during an air break is faster than N₂ washout during 100% O₂ PB due to the release of the vasoconstrictive action of high O₂ partial pressure.

Computing Theoretical Tissue N₂ Pressure for Decompression Dose Model

$P_i N_2 = P_0 N_2 + (P_a N_{2i} - P_0 N_2) \cdot (1 - \exp(-k_i \cdot t_i))$, where $P_0 N_2$ is initial equilibrium tissue N₂ pressure taken as 11.6 psia at sea level, $P_a N_{2i}$ is breathing mixture partial pressure of N₂ over the i^{th} time interval during the PB, t_i is in min, and pressure is psia.

$k_i = ((\ln 2 / t_{1/2base}) \cdot (0.39 \cdot P_a N_{2i} + 0.9))$, where $k = 0.011$ ($t_{1/2}$ is 60 min) when $P_a N_2 = 0$ psia and 0.069 ($t_{1/2}$ is 10 min) when $P_a N_2 = 11.6$ psia, with $t_{1/2base} = 54$ min.

Computing P(DCS) for Altitude Exposure of 4.37 psia

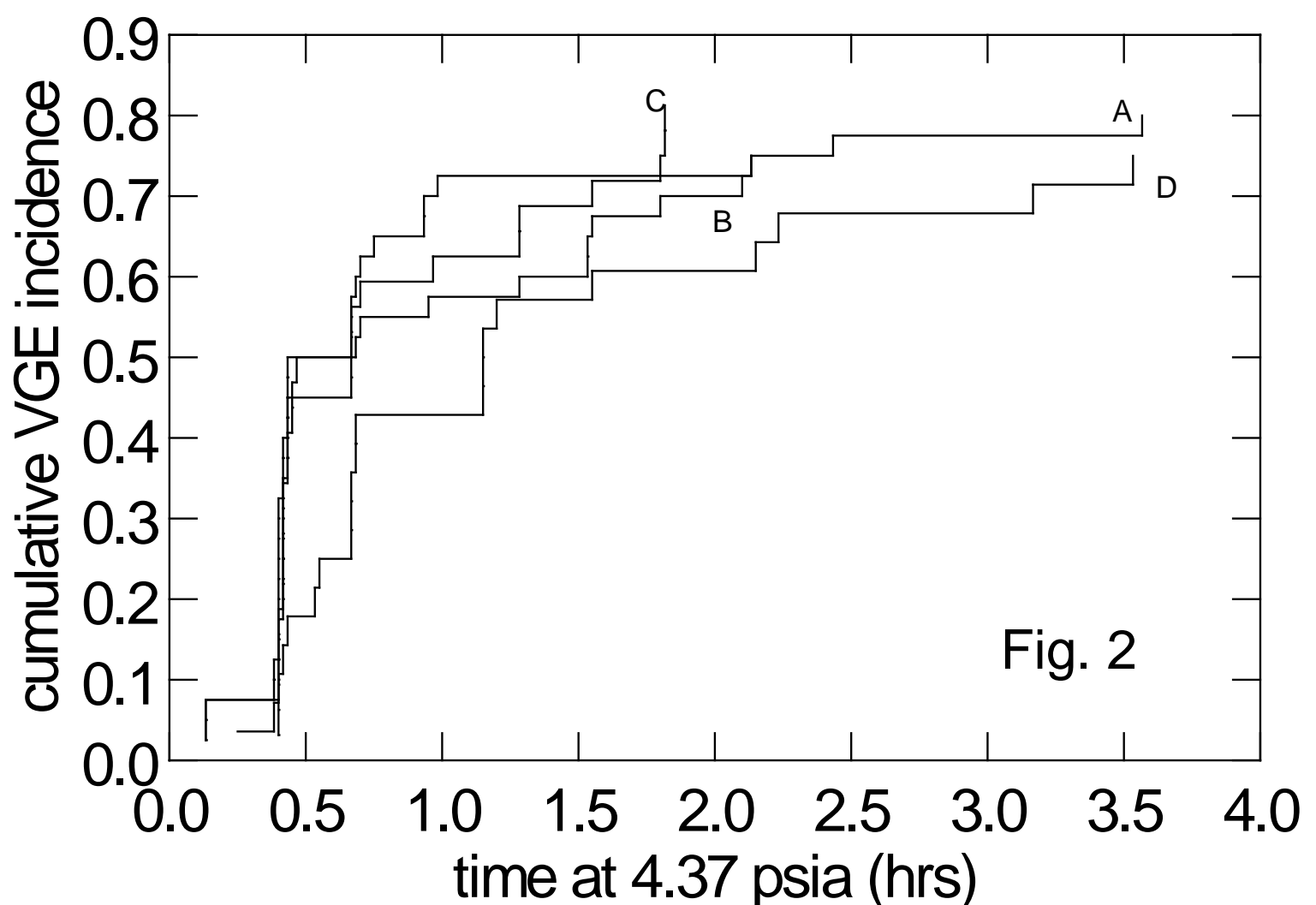
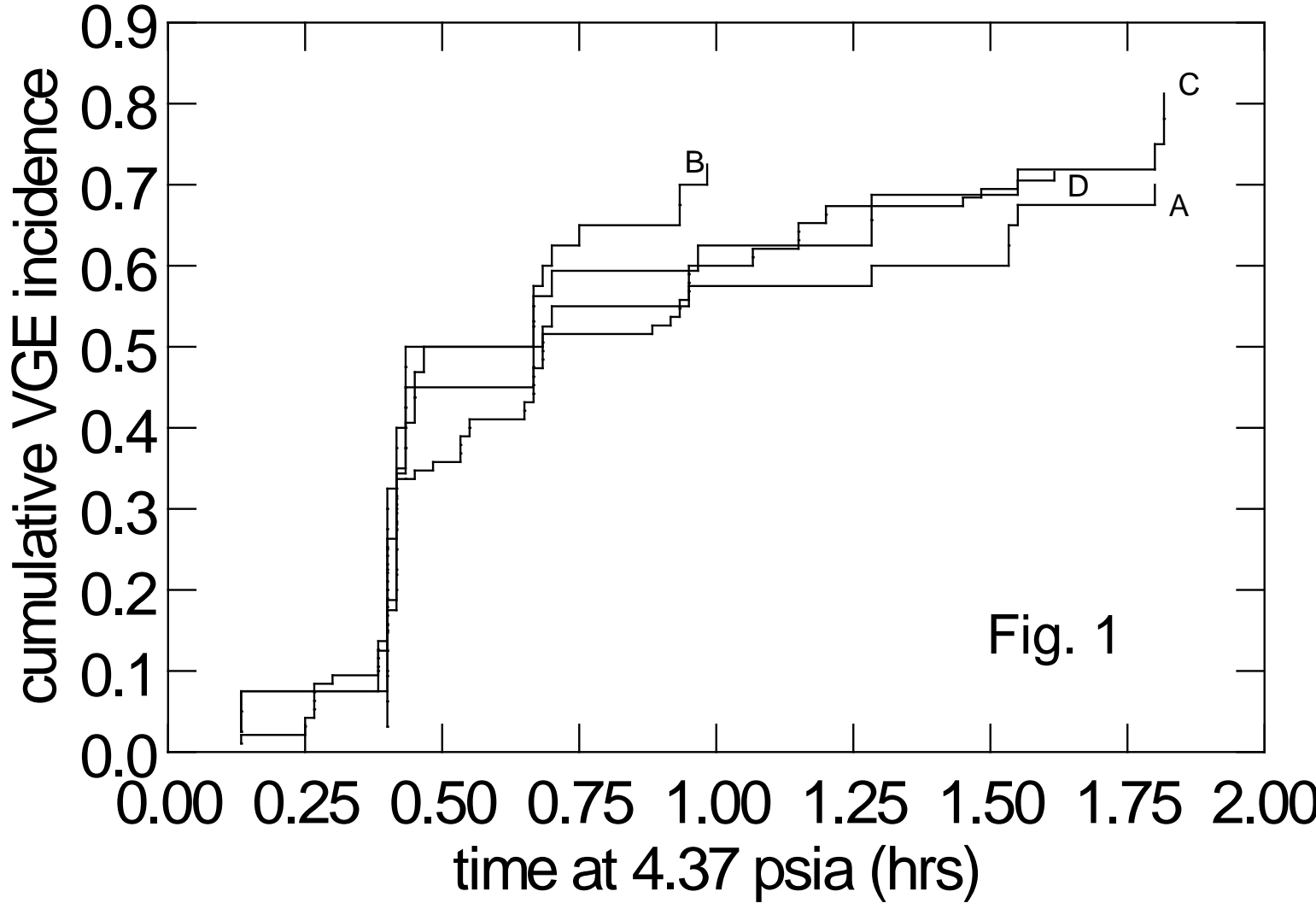
$P(DCS)_t = 1 - \exp(-\ln[1 + (P_i N_2 - 4.37)^x \cdot (t \cdot \beta)^y])$, where t is in hr.

SYSTAT (ver.8) used to compute α , β , and χ coefficients in the accelerated log logistic survival model based on recorded survival times influenced by the PB and exposure conditions of the tests.

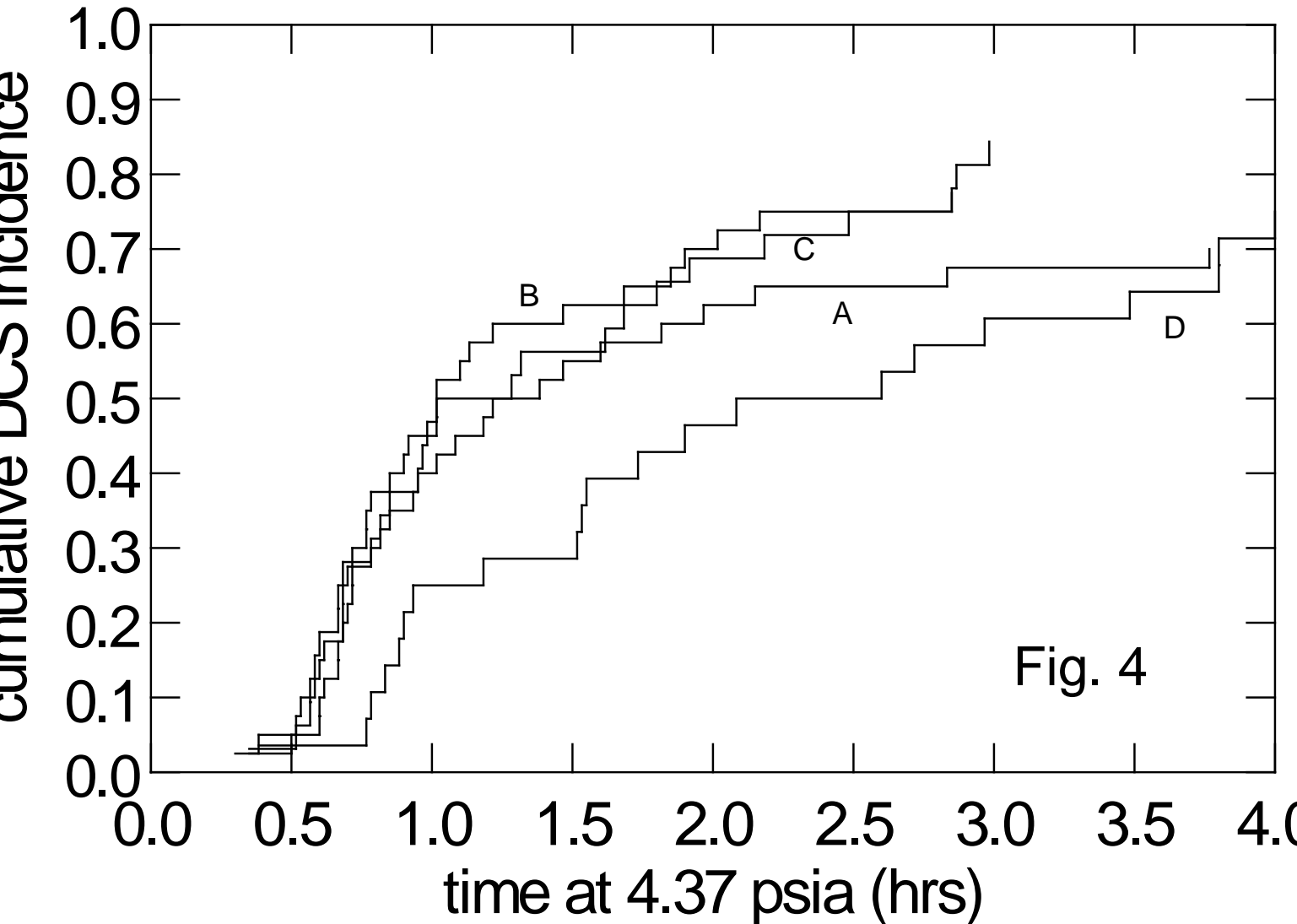
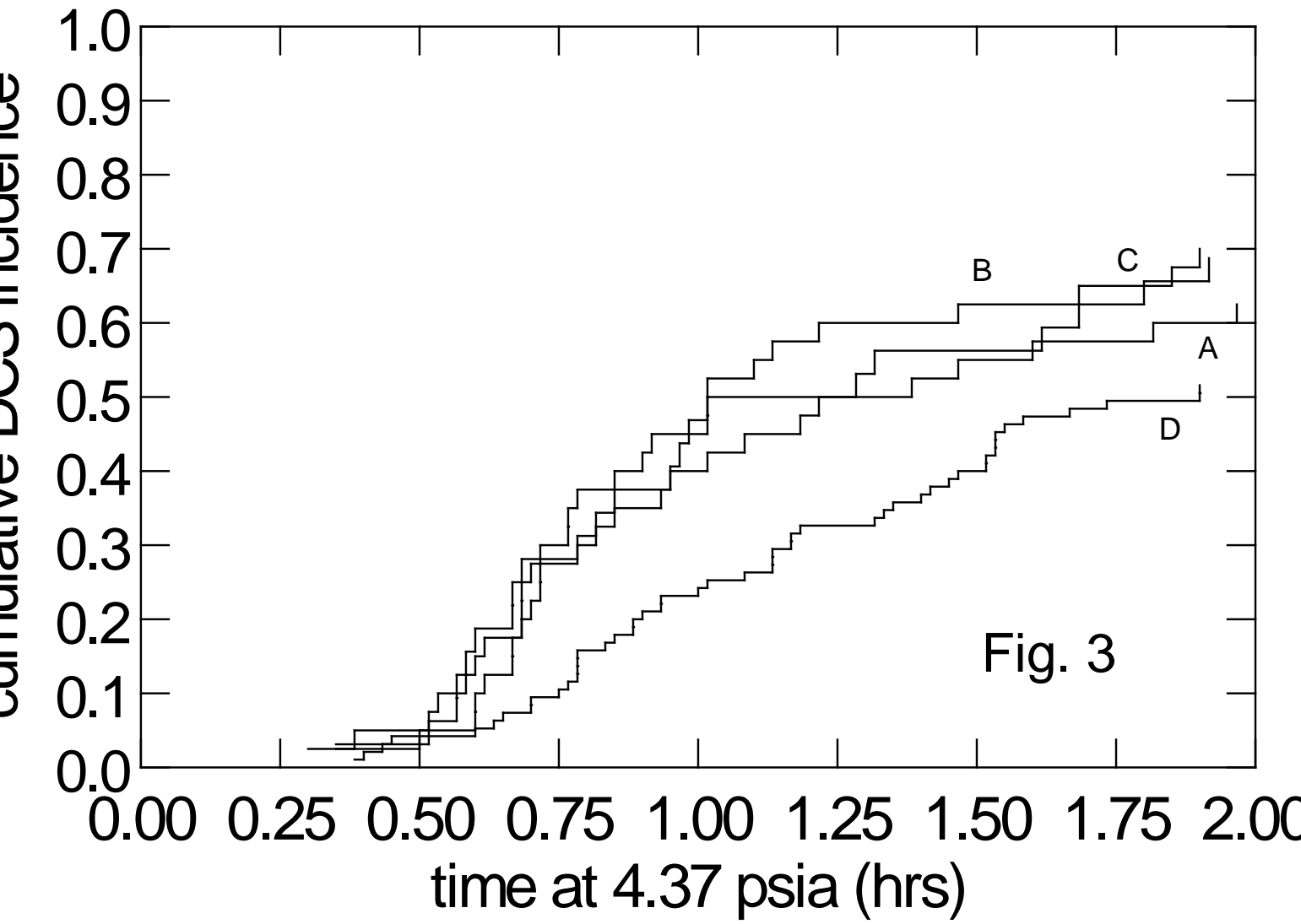
Legend for curves to follow:

- A = 10-min air break at 30 min into a 60 min 100% O₂ PB
- B = 20-min air break at 30 min into a 60 min 100% O₂ PB
- C = 60-min air break at 30 min into a 60 min 100% O₂ PB
- D = 60 min 100% O₂ PB, control data

RESULTS



Figs. 1 and 2 show no difference in the pattern of cumulative VGE incidence for the three curves for air breaks, so we conclude that a 10, 20, and even 60 min air break 30 min into a 60 min PB produce VGE failure times that are statistically indistinguishable. This conclusion extends to a comparison with the VGE failure times in the controls. In both the two-hr and four-hr matched exposure time data, and accounting for interval censoring, the p-values from the Tarone-Ware log-rank test for any comparison of control and air break duration VGE data was > 0.14 (8). Note the early onset and rapid increase in cumulative VGE incidence in both the control and air break data.



Figs. 3 and 4 show no difference in the pattern of cumulative DCS incidence for the three curves for air breaks, so we conclude that a 10, 20, and even 60 min air break 30 min into a 60 min PB produce DCS failure times that are statistically indistinguishable. But there is an effect of air break on the pattern of DCS failure times when referenced to the controls. The Kaplan-Meier estimate of the survival function accounting for censored observations stratified by air break times is evaluated with the Tarone-Ware log-rank test. The 20-min and 60-min air break DCS failure times in our two-hr matched exposure time data are different than the control DCS failure times (p<0.03). And the 20-min and 60-min air break DCS failure times in our four-hr matched exposure time data are near to statistical significance than the control DCS failure times at p<0.06 (8).

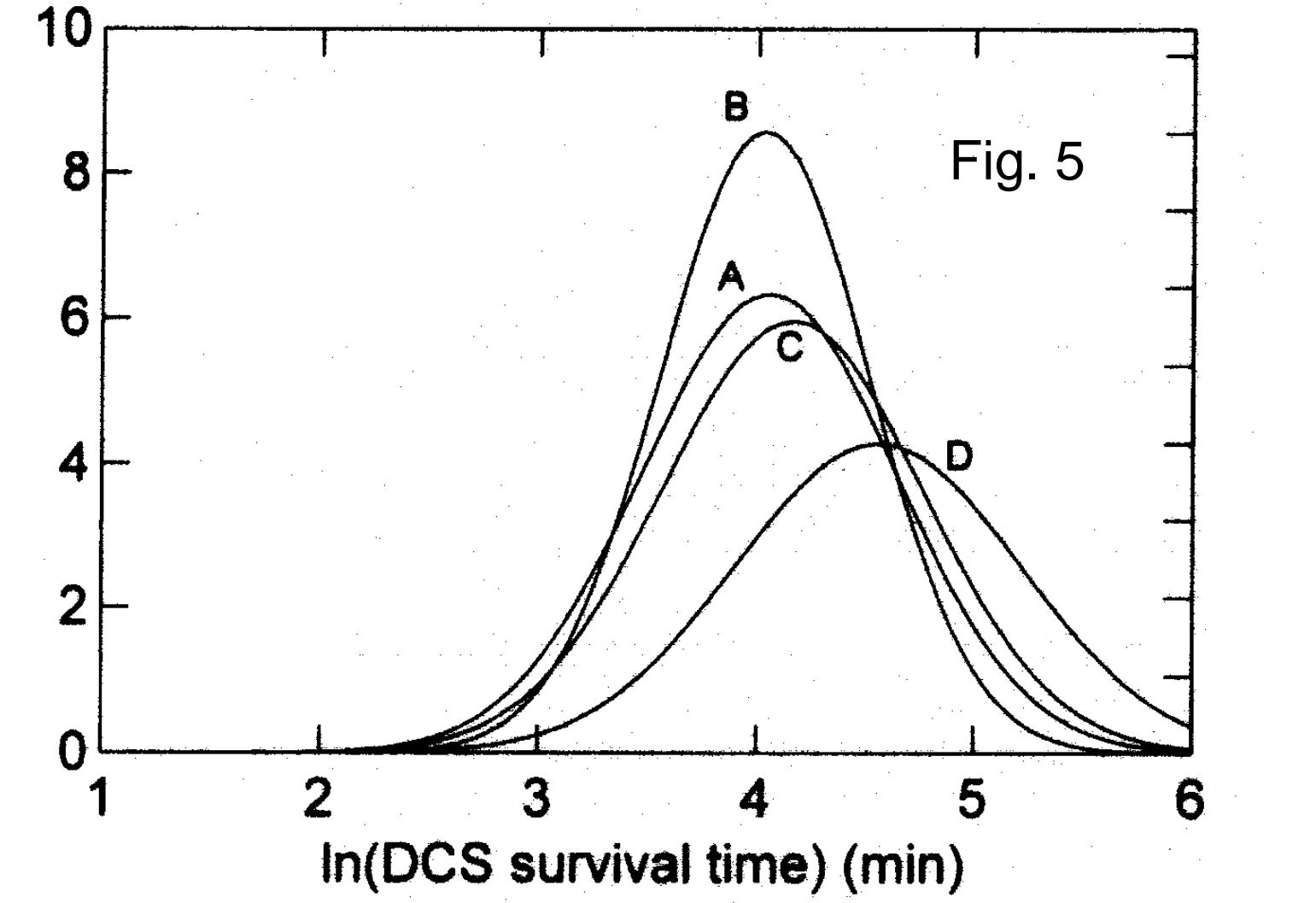
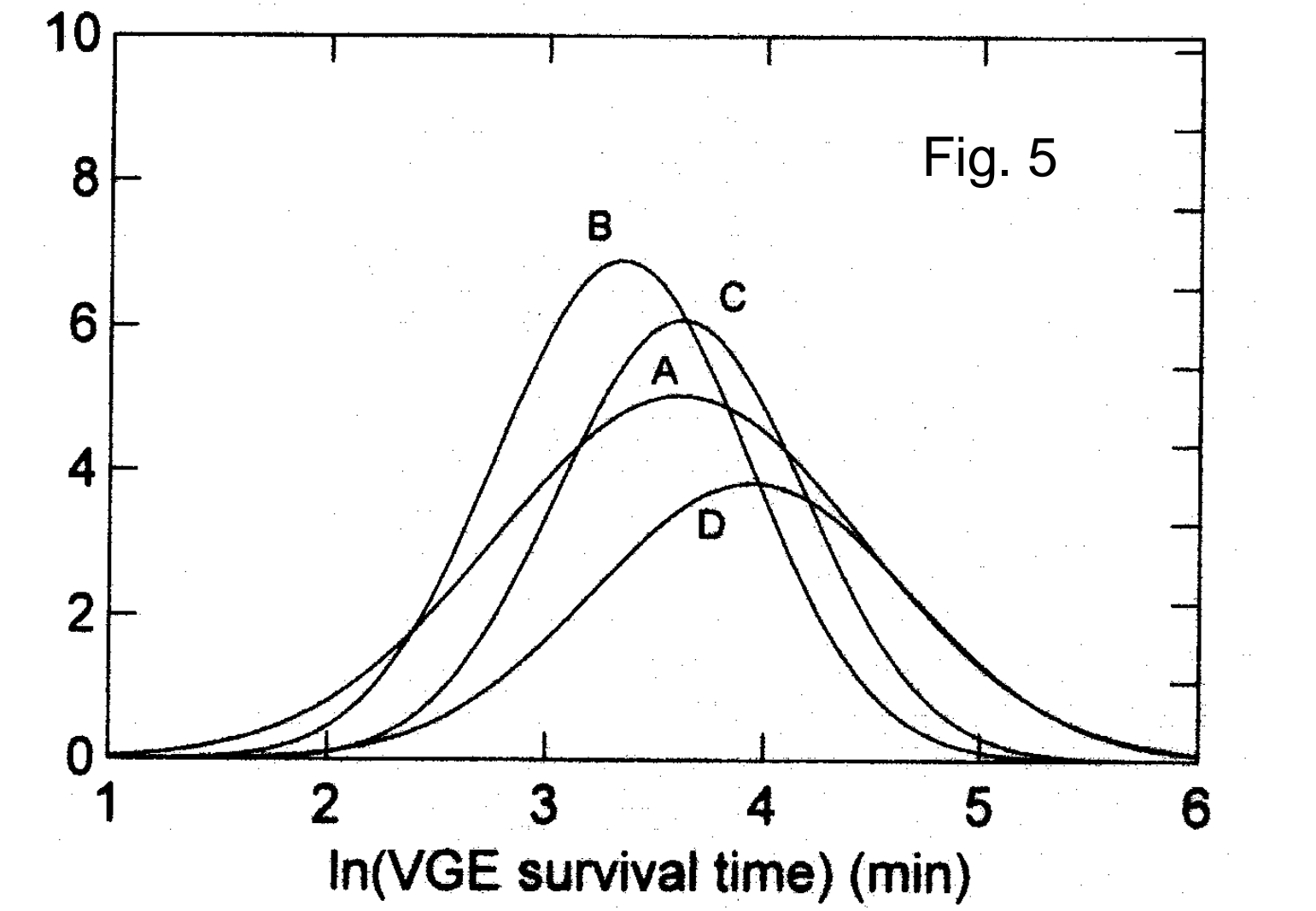


Fig. 5. Comparison of normal density plots for ln(VGE) and ln(DCS) survival times in the 4-hr exposure data (28 controls and 112 air breaks). Notice the early onsets and mean ln(VGE) times compared to the later onsets and mean ln(DCS) times. Air breaks in PB did not change the indirect association between VGE first detected in the right heart circulation)and symptom onset. 15% of those with DCS had no VGE (21/140), 16% with VGE had no DCS (23/140), 7% had no DCS or VGE (10/140), and 61% had DCS and VGE (86/140).

TABLE 1. MODEL RESULTS TO DESCRIBE DCS SURVIVAL TIMES

model	LL, p – value*	tested hypothesis
log logistic survival	-260.52	no variable accounts for the survival times, computer fits α and β
accelerated log logistic survival: $P_i N_2 - 4.37)^x$	-----	variables account for the survival times, computer fits α , β , and χ pressure difference to a power as decompression dose
$P_i N_2 f(360 t_{1/2})$	-259.75, p = 0.21	symmetrical, long half-time kinetics to describe DCS outcome
$P_i N_2 f(60 t_{1/2})$	-257.66, p = 0.016	symmetrical, short half-time kinetics describe DCS outcome
$P_i N_2 f(60 t_{1/2} + 10 t_{1/2})$	-257.05, p = 0.008	asymmetrical, short half-time for washout and even shorter for washin to describe DCS outcome
$P_i N_2 f(360 t_{1/2} + 36 t_{1/2})$	-257.79, p = 0.019	asymmetrical, long half-time for washout and very short for washin to describe DCS outcome

LL is computed log likelihood from survival analysis regression, p – value is from Likelihood Ratio Test where p < 0.05 indicates improvement in the model over the baseline model with LL -260.52.

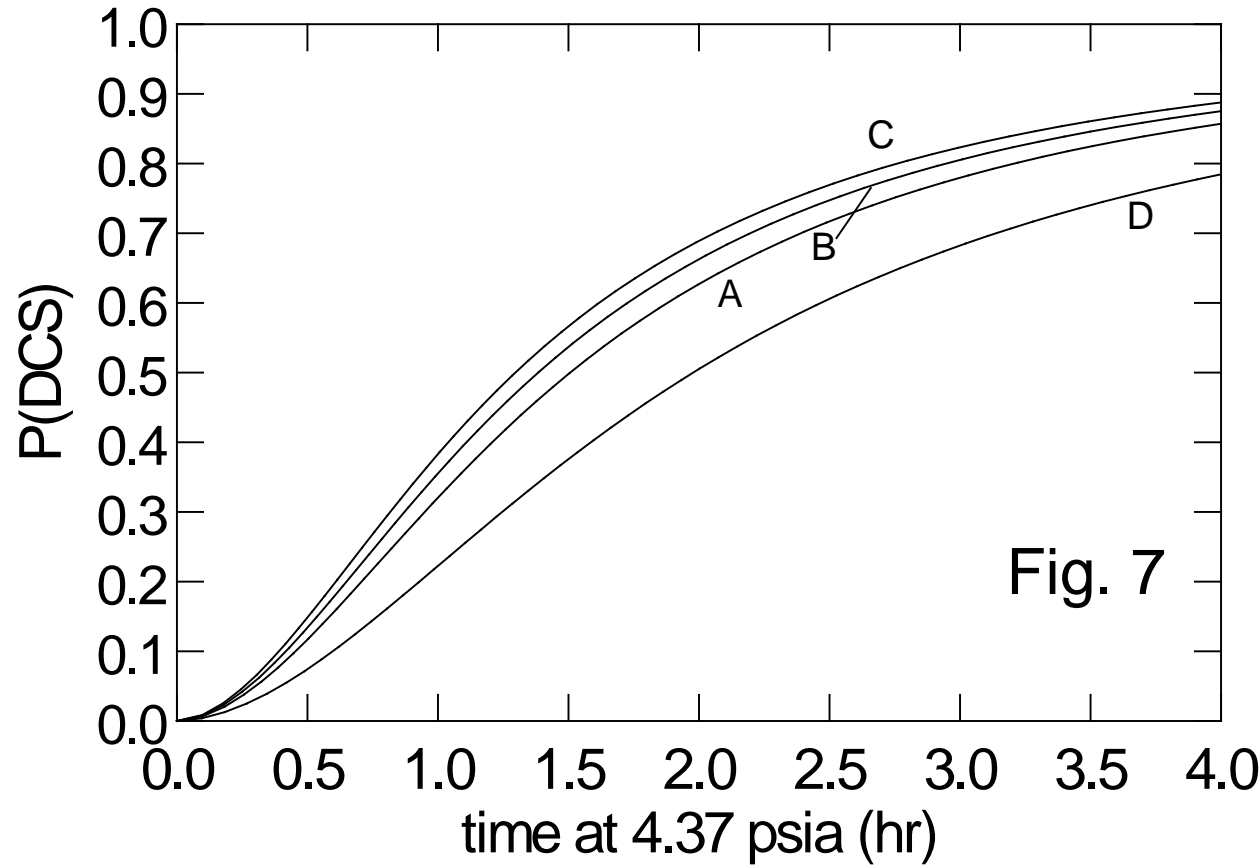


Fig. 7 Combined 95 controls and 112 air breaks into one predictive survival model: $P(DCS)_t = 1 - \exp(-\ln[1 + (P_i N_2 - 4.37)^{0.652} \cdot (t \cdot 0.515)^{1.835}])$. Mean $P_i N_2$ for curve A = 6.413 psia, 6.964 for B, 7.486 for C, and 5.320 for D.

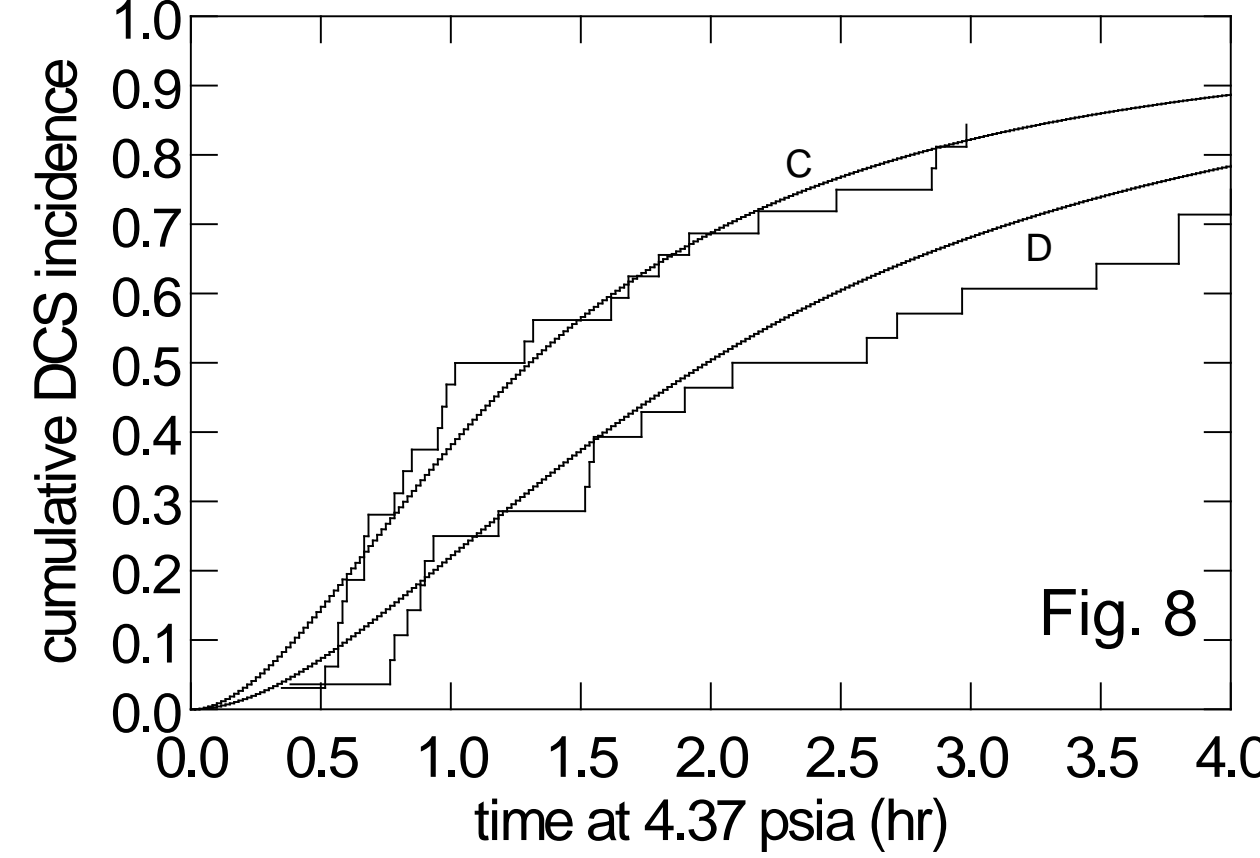


Fig. 8 Goodness-of-fit is reasonable, predicted and observed survival times for curve C have p = 0.48, and p = 0.15 for curve D. Curves A and B (not shown) have p = 0.69, where p > 0.05 indicates prediction is not significantly different than observation (9).

CONCLUSIONS / DISCUSSION

A N₂ kinetic model that maximized the small differences in these data required very short half-times. This is a perplexing conclusion since very high DCS incidence is associated with very low computed tissue N₂ pressure, so the model is incomplete, but utilitarian over a narrow range of test conditions.

If returning to the control condition is an acceptable compensation for an air break in a 60 min PB, then our regression model is a quantitative way to define O₂ payback time.

TABLE 3. COMPUTED O₂ PAYBACK TIMES

duration of air break (min) \Rightarrow table not validated

	5	10	15	20	25	30
5						
10		5		8		9
15						
20		11		16		18
25						
30		17		24		27
35						
40		22		31		35
45						
50		29		40		45
55						

Add the additional O₂ PB in the table to the balance of the required 60 min PB. Select the next lowest PB time and the next highest air break time if actual times are between the table values. The times in the table assume a minimum of six min on 100% O₂ elapses during the ascent to 4.37 psia – chart specific to Brooks AFB data.

Our approach to compute O₂ payback time is not appropriate outside the range of tested conditions.

The control data are from an "effective PB" of 66 min, and our model computes an effective PB of 50 min for the 10-min air break, 43 min for the 20-min air break, and 37 min for the 60-min air break.

Our regression model has not been prospectively validated, *so our conclusions are hypotheses rather than operational recommendations.*

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